The Assessment of Cases for Mitral Valvotomy and the Results of Operation

By J. F. PANTRIDGE, M.C., M.D., M.R.C.P.(LOND.)
Physician, Royal Victoria Hospital
T. B. SMILEY, M.C., F.R.C.S., and E. W. HENRY, M.D.
Thoracic Surgeon Registrar

IN certain cases of inactive rheumatic heart disease when the lesion is predominantly mitral stenosis dramatic results may be obtained from surgical relief of the valvular obstruction (Bailey, 1949; Glover, et al., 1950; Baker, et al., 1950, 1952). Unfortunately, not all cases of mitral stenosis will benefit from operative interference. The major difficulty associated with the surgical treatment of rheumatic heart disease is that of deciding in which case, and at what stage in the course of the disease, operative interference is required. The responsibility for this decision is borne by the physician. In some cases it will be easy to determine whether or not benefit will be obtained from operation; in others it may be a matter of considerable difficulty.

The object of this communication is to discuss the selection of patients for mitral valvotomy and to report 121 cases, 105 of whom were selected for this operation by one of us (J.F.P.).

Mitral valvotomy is obviously indicated if a patient is becoming progressively incapacitated and the condition is one of "pure" mitral stenosis, i.e., mitral stenosis uncomplicated by significant mitral incompetence or aortic valvular involvement.

It is apparent that by no means all patients with "pure" mitral stenosis are sufficiently incapacitated to require operation. The proper selection of cases requires therefore :—

(a) Information regarding the effects of mitral valvular obstruction on the circulation.

(b) A clinical classification of patients suffering from the disease.

THE PULMONARY CIRCULATION IN MITRAL STENOSIS.

The effect of mitral stenosis on the circulation may be predicted from simple hydraulic principles. When an orifice is narrowed an increase in the pressure gradient across this orifice will be required if the rate of flow is to be maintained, and any increase in the rate of flow will require a further increase in the pressure gradient. Mitral stenosis is associated with a rise in left auricular pressure and with further elevation of this pressure when blood flow is increased as by exertion.

The rise in pressure in the left auricle is necessarily followed by a rise in pulmonary vascular pressure. The pressure in the venous side of the pulmonary circulation may be measured by introducing a cardiac catheter into one of the pulmonary arteries and advancing it till it is made to block a small pulmonary

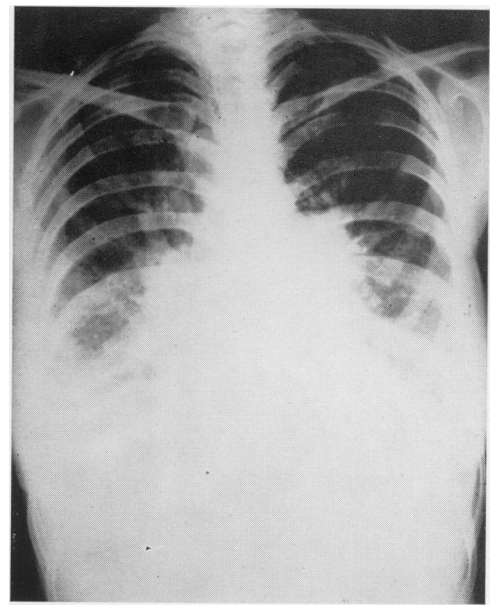

Fig. 1

Mitral stenosis in pregnancy. Chest film before operation showing gross pulmonary congestion.

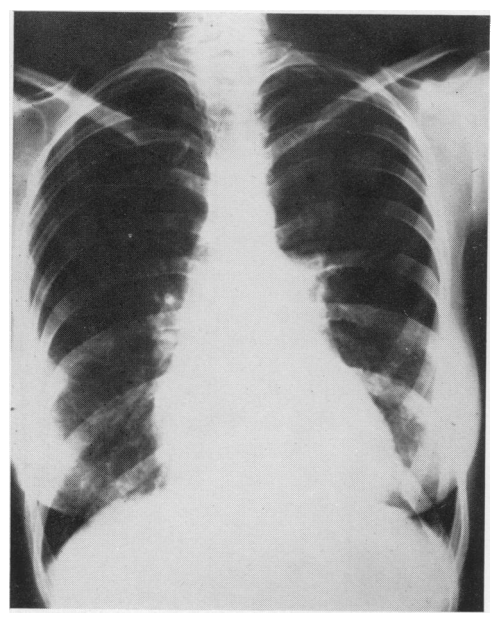

Fig. 2

Chest film after operation. Hilar vascularity is almost normal.

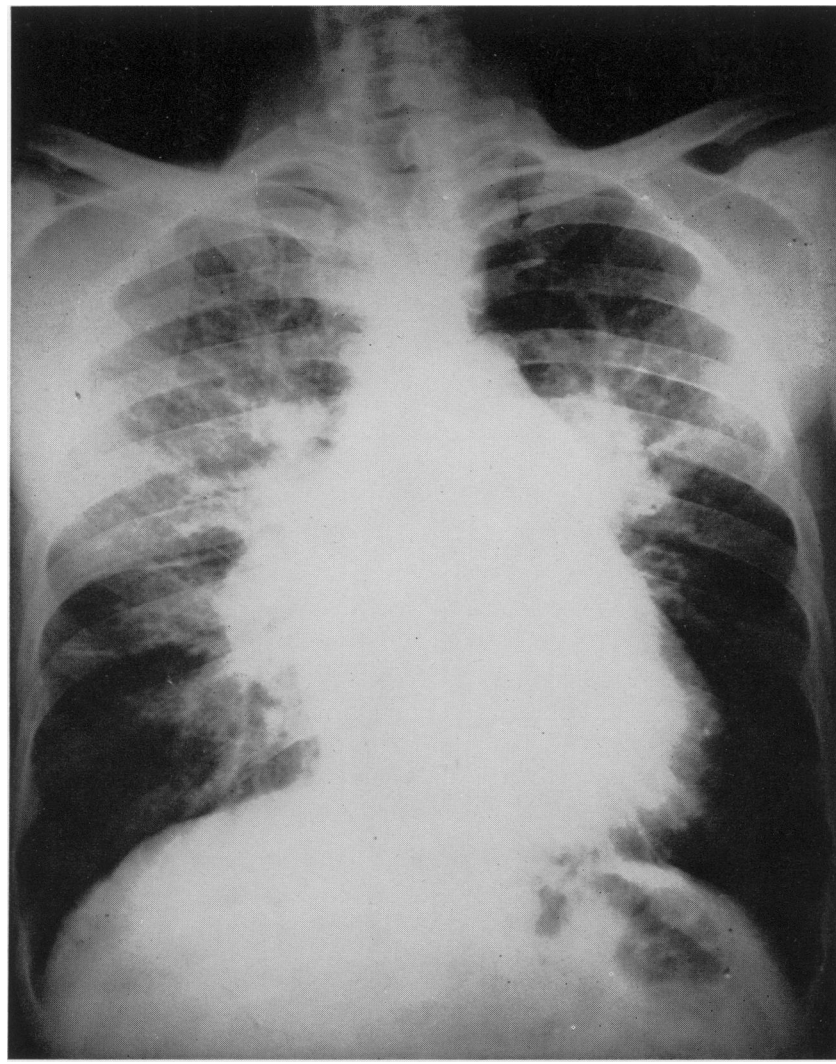

Fig. 3

W. G. (Case 49). Chest film before operation showing gross pulmonary engorgement.

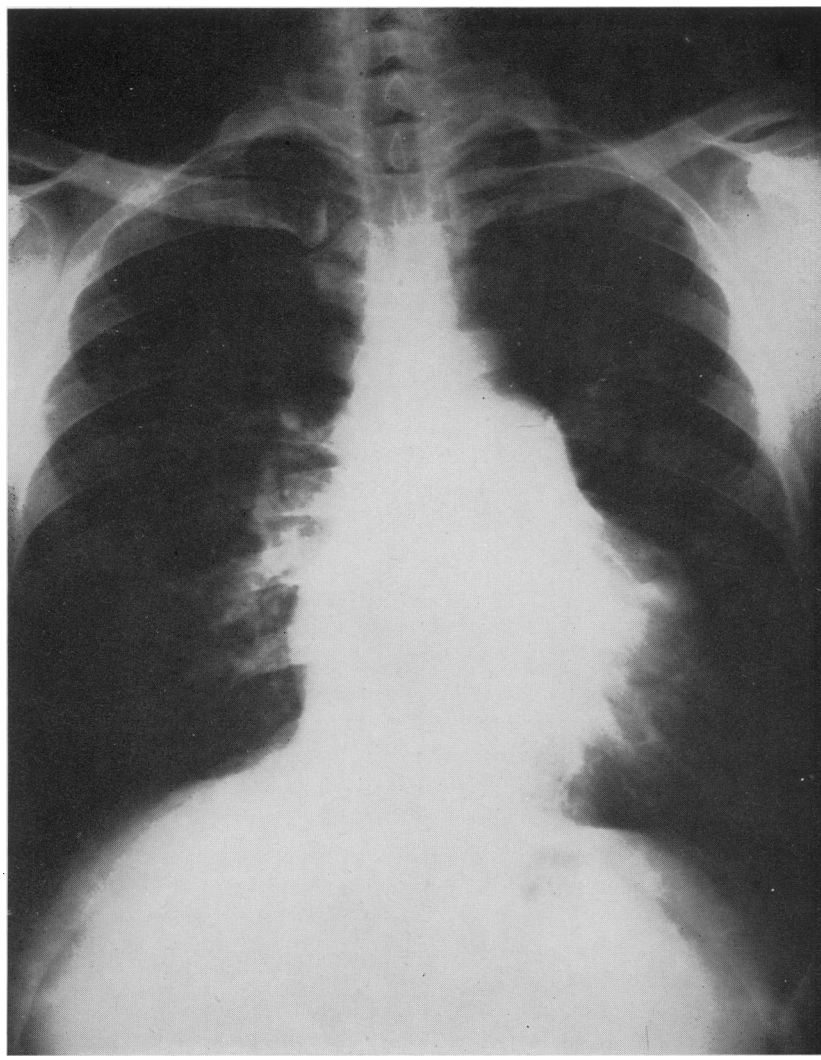

Fig. 4

W. G. (Case 49). Chest film after operation : lung fields are clear. Cardiac outline shows little change.

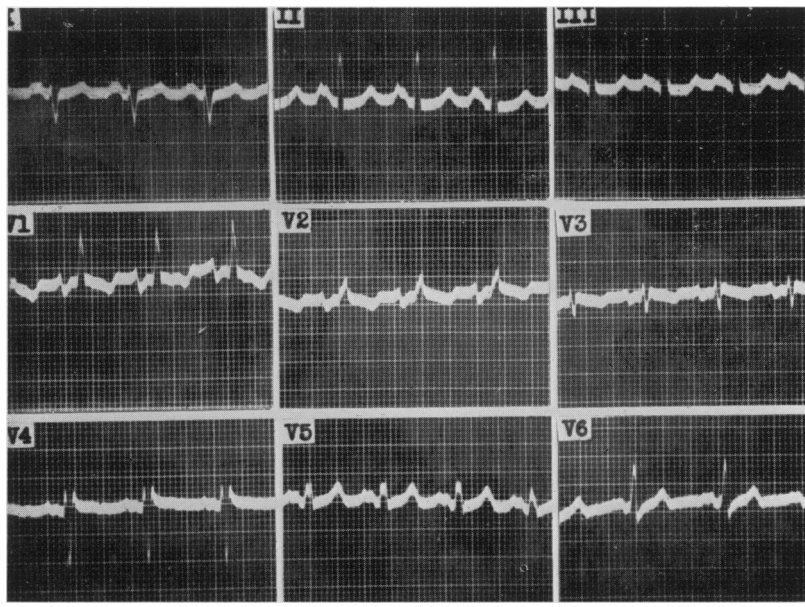

Fig. 5

A. C. (Case 68). Pre-operative E.C.G. shows gross right ventricular hypertrophy.

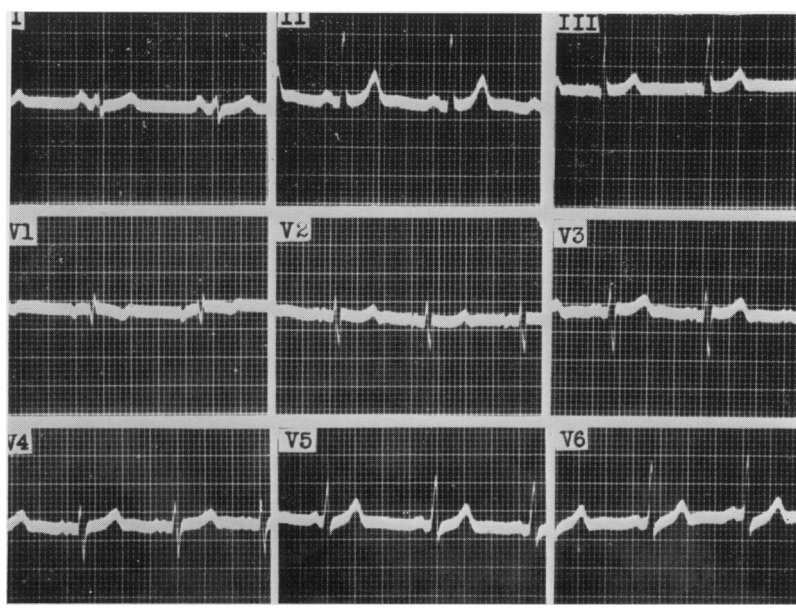

Fig. 6

A. C. (Case 68). Post-operative E.C.G. shows that the signs of right ventricular hypertrophy have entirely gone.

vessel (Hellems, et al., 1948; Lagerlof and Werko, 1949). It is probable that the pressure recorded at this point, the pulmonary capillary venous pressure, closely approximates the left auricular pressure (Dow and Gorlin, 1950; Epps and Adler, 1953). Pulmonary œdema is likely to occur when the pulmonary capillary venous pressure exceeds the plasma colloid osmotic pressure (30 mm. Hg.). Gorlin and Gorlin (1951) placed the pulmonary œdema threshold at 35 mm. Hg. and indicated the close relationship which must exist between the mitral valve area, the rate of blood flow through the valve, and the pulmonary capillary venous pressure. Thus when the valve area is normal (4 to 6 cm^2.), the rate of blood flow may be increased to as much as 700 cc. per sec. (adequate for the most strenuous activity), without significant rise in the left auricular or pulmonary capillary venous pressure. When the area is narrowed to 2.5 cm^2. flow rates exceeding 400 cc. (required by strenuous activity) will cause a rise of pulmonary capillary venous pressure to 35 mm. of Hg. or more and therefore give rise to pulmonary œdema. Flow rates exceeding 250 cc. per sec. cannot be tolerated if the area is reduced to 1.5 cm^2. When the area is less than 1 cm^2. flow rates of 150 cc. cannot be maintained without a rise of pulmonary capillary venous pressure above 35 mm. Hg. Patients with severe mitral stenosis (valve area 1 cm^2. or less) are therefore likely to have pulmonary œdema at rest.

A rise in pulmonary venous pressure is followed by a rise in pulmonary artery pressure. However, in some 25 per cent. of cases of mitral stenosis the rise in pulmonary artery pressure is disproportionately great. This disproportionate rise in pulmonary artery pressure is due to increased pulmonary arteriolar resistance. How much of this increase in arteriolar resistance is due to structural changes in the arteriolar wall and how much to vasoconstriction is not known. Its effect is to prevent a marked rise in pulmonary venous pressure and hence to "protect" the patient from pulmonary œdema.

Despite the fact that in some circumstances the protective increase in pulmonary vascular resistance may temporarily fail permitting flooding of the capillaries and pulmonary œdema, patients with mitral stenosis may, on the basis of the presence or absence of an increase in pulmonary vascular resistance, be classified as "protected" or "unprotected." Unprotected mitral stenosis is associated with symptoms and signs referable to pulmonary venous engorgement—hæmoptysis, nocturnal dyspnœa, and recurrent attacks of pulmonary œdema. Patients with "protected" mitral stenosis tend to avoid these manifestations of pulmonary congestion but may be no less disabled since the circulatory obstruction in the pulmonary arterioles results in their inability to increase the cardiac output on activity. "Protected" mitral stenosis gives rise to clinical, radiological, and sometimes electrocardiographic evidence of right ventricular hypertrophy. Eventually congestive heart failure results from failure of the hypertrophied right ventricle.

Clinical Classification of Patients with Mitral Stenosis.

A suggested classification is :—

Group 1.—Asymptomatic mitral stenosis. The characteristic mitral diastolic

murmur is present but disability is negligible. Rise in pulmonary vascular pressure is minimal.

Group 2.—The disability is moderate but not increasing. Patients are able to do their ordinary duties without discomfort. Cardiac catheterization shows a slight rise in pulmonary vascular pressure at rest and a moderate rise on exertion.

Group 3.—There is evidence of increasing disability. "Unprotected" patients in this group will have symptoms and signs of pulmonary congestion, history of hæmoptysis, marked diminution in exercise tolerance, perhaps nocturnal dyspnœa, and moist sounds at the lung bases. Cardiac catheterization will show a considerable rise in pulmonary capillary venous pressure without marked rise in the pulmonary artery pressure. "Protected" patients in this group will have gross impairment of exercise tolerance due to a low fixed cardiac output. There will be clinical evidence of right ventricular hypertrophy, prominent "a" waves in the jugular pulse, a heaving right ventricular outflow tract felt to left of the sternum, the Graham Steele murmur of pulmonary incompetence, and accentuation of the second component of the split pulmonary second sound. X-ray screening will confirm the presence of right ventricular enlargement and show a prominent pulmonary conus. The electrocardiogram may show a præcordial lead pattern characteristic of right ventricular hypertrophy. Cardiac catheterization will show a marked increase in the gradient between the pulmonary artery pressure and the pulmonary capillary venous pressure. When first seen, "protected" patients in this group may show signs of early right ventricular failure.

Group 4.—Patients in this group are completely incapacitated because of intractable pulmonary congestion or intractable right heart failure.

Cases in group 1 obviously do not require surgical intervention. Cases in group 2 may require operation if the degree of static disability is great. It has, however, been our policy to defer operation when it is entirely clear that the disability is not progressive. The possibility of improvement in operative technique cannot be overlooked, so that much may be gained by waiting a few years. The condition of patients in this group is, however, carefully reviewed at three to six monthly intervals to ensure that the earliest evidence of progression of the disease is recognised. When doubt exists as to whether a patient should be placed in group 2 or 3, the pulmonary vascular pressures recorded by cardiac catheterization will be decisive. Groups 3 and 4 are those which, in the absence of contraindications, are selected for operation.

Contraindications to Operation.

1. Active infection, due either to active rheumatism or subacute bacterial endocarditis, is an obvious contraindication to operation. Unfortunately it is impossible, with the clinical and laboratory methods at present available, to exclude with certainty smouldering rheumatic activity. Inquiry regarding recent joint or limb pains, electrocardiographic investigation, erythrocyte sedimentation rate, and white cell count may give no indication of rheumatic activity, and yet Aschoff nodules may be found in the biopsy of the left auricular appendage removed at operation

(McKeown, 1953). It has been shown, however, that the incidence of unsuspected rheumatic activity, which in some series has been as high as 45 per cent., progressively declines with age, being 89 per cent. in the age group 20-25 and 14 per cent. in the age group 45-50 (McNeely, et al., 1953).

2. Aortic valvular involvement, if associated with clinical, radiological or electrocardiographic evidence of left ventricular enlargement, is, as a rule, a contraindication to mitral valvotomy. An early diastolic murmur heard along the left side of the sternum in patients with mitral stenosis and marked pulmonary hypertension—the Graham Steele murmur of pulmonary incompetence—should not be confused with the murmur of aortic incompetence. An aortic diastolic murmur associated with the peripheral signs of aortic reflux—a Corrigan pulse—and raised pulse pressure is an unfavourable sign. However, if the patient's condition is precarious because of the pulmonary congestive features associated with tight mitral stenosis, aortic valvular involvement, unless obviously very considerable, is not regarded as a bar to operation.

A double aortic murmur was present in nine cases (7.5 per cent.) in this series. In two of these it was associated with the peripheral signs of aortic incompetence and with slight to moderate left ventricular enlargement. In both improvement after mitral valvotomy has been remarkable. Two cases with a double aortic murmur, but without change in the peripheral pulse or left ventricular enlargement, failed to improve following operation. In neither of these can the poor result be attributed to the aortic lesion.

3. Predominant mitral incompetence is a contraindication to the present operation of mitral valvotomy. Mitral stenosis, complicated by slight to moderate incompetence, may, however, be successfully dealt with by operation. It may, in fact, be noted that the amount of mitral reflux diminishes when the adherent commissures are freed.

The great difficulty in the selection of patients for valvotomy is to predict the condition of the valve when the signs produced by the mitral lesion are neither those of "pure" stenosis or "pure" incompetence. In this connection it is of value to keep in mind the signs produced by "pure" stenosis as opposed to those produced by "pure" incompetence.

The characteristic findings in "pure" mitral stenosis are a low-pitched mitral diastolic murmur, maximal in presystole and ending in a slapping first heart sound, the absence of a murmur in systole and the presence of a third heart sound in early diastole, the opening snap of the stenosed valve. This opening snap is maximal inside the apex, but when loud may be widely conducted. It is high-pitched, of short duration, and closely resembles in character the normal second heart sound. It is best heard in expiration. It is to be distinguished from the second component of the split pulmonary second sound which is accentuated in the presence of pulmonary hypertension and is best heard in the pulmonary area. Splitting of the pulmonary second sound is maximal during inspiration. When the condition is "pure" mitral stenosis there will be no clinical, radiological, or

electrocardiographic evidence of left ventricular enlargement. However, in some cases of protected stenosis with marked pulmonary hypertension displacement of the left ventricle by the grossly enlarged right ventricle may result in erroneous radiological evidence of left ventricular enlargement. In these cases the electrocardiogram is of great value, since it will usually show a right ventricular hypertrophy pattern in the præcordial leads with displacement of the transitional zone towards the left.

Pure mitral incompetence is usually associated with a loud mitral systolic murmur often maximal in late systole and conducted to the axilla or as far as the left scapula and with absence of accentuation of the mitral first heart sound. A third heart sound is frequently heard in diastole, and differs from the opening snap of the mitral valve in that it is later in diastole, is of longer duration, of lower pitch, and is confined to the apex. It is thought to be due to rapid ventricular filling (Brigden and Leathem, 1953). A mid-diastolic murmur may be audible. There will be radiological and in many cases electrocardiographic evidence of left ventricular enlargement. X-ray screening will usually show obvious systolic expansion of the left auricle in both right oblique and antero-posterior views.

When the character of the mitral valvular lesion is in doubt, the signs of stenosis are weighed against those of incompetence as indicated in Table I.

TABLE I.

RECOGNITION OF MITRAL STENOSIS AND INCOMPETENCE.

SIGN.	STENOSIS.	INCOMPETENOE.
Accentuated or slapping mitral first heart sound.	Usually present.	Uncommon.
Mitral opening snap.	Common.	Rarely, if ever, occurs.
Mitral systolic murmur.	Not usually loud or notably conducted.	Often loud and conducted to axilla.
Radiological evidence of		
(a) left ventricular enlargement.	Absent unless there is a complicating condition, e.g., hypertension or an aortic valvular lesion.	Left ventricular enlargement usually evident.
(b) left auricular systole expansion on screening.	Backward movement of barium filled œsophagus may be present in right oblique position.	Systolic expansion usually present in both oblique and antero-posterior views.
Electrocardiogram.	May show evidence of right ventricular enlargement.	May show evidence of left ventricular enlargement.
Pulmonary vascular pressure determined by cardiac catheterization.	Marked pulmonary hypertension common.	Marked pulmonary hypertension distinctly uncommon.

It was found that if the first heart sound was slapping, the opening snap audible, and left ventricular enlargement absent, the mitral lesion was predominantly stenosis, irrespective of the character of the mitral systolic murmur.

However, the accentuation of the first heart sound and the opening snap may both be abolished by calcification of the mitral valve. Absence of these features is not therefore conclusive evidence of mitral incompetence. Further, when right ventricular enlargement is considerable determination of the size of the left ventricle may in some cases be difficult.

For these reasons great care must be taken to avoid an erroneous diagnosis of predominant incompetence and hence to refuse the patient the chance of benefit from operation. If this possibility is constantly in the physician's mind the reverse mistake will occasionally be made and an unexpected degree of mitral incompetence may be discovered at operation.

Predominant mitral incompetence was found at operation in five of the 121 cases in this series. It was present in two of 105 cases assessed by one of us (J.F.P.).

The first of these two patients (E.M.) showed a mitral diastolic murmur, slapping mitral first heart sound, and a grade III mitral systolic murmur conducted to the axilla. The mitral opening snap was absent. There was slight left ventricular enlargement on X-ray screening.

The second case (A.M.), a male aged 43 years, was first seen nine months before operation because of hæmoptyses. There was a mitral diastolic murmur but no accentuation of the mitral first heart sound and no mitral opening snap. There was no mitral systolic murmur. An aortic diastolic murmur was audible but the peripheral pulse and pulse pressure were normal. X-ray screening showed gross right ventricular enlargement and apparent considerable enlargement of the left ventricle. The præcordial leads of the electrocardiogram showed a right ventricular hypertrophy pattern with shift of the transitional zone to the left as far as V 6. The radiological appearance of left ventricular enlargement was therefore thought to be more apparent than real. The electrocardiogram in this case is of interest since it has been stated that a right ventricular hypertrophy pattern in mitral valvular disease is always associated with predominant stenosis (Frazer and Turner, 1953).

It is true that both these cases were regarded as unsuitable for operation at the initial assessment. Subsequently operation was agreed to in the hope that sufficient stenosis was present to enable operative interference to arrest the rapid downhill course. Needless to say, we hold that if there is doubt about the relative degree of stenosis and incompetence and the patient is pursuing a rapidly downhill course it is better to risk the possibility of a useless operation than to deny the patient the chance, however remote, of benefit from surgery.

Factors Increasing Operative Mortality.

1. The presence of auricular fibrillation undoubtedly increases the mortality but is certainly not a contraindication to operation. The risk is that of systemic embolism from dislodged auricular thrombi. A history of or evidence of previous

systemic emboli is now regarded by some as an indication for mitral valvotomy since removal of the left auricular appendage and decrease in the pooling of blood in the left auricle diminish the tendency toward the formation of left auricular thrombi (Griffiths, et al., 1953). It is of interest that unsuspected rheumatic activity is less common when auricular fibrillation is present (McNeely, et al., 1953).

2. Extreme severity of the effects of mitral obstruction does not contraindicate mitral valvotomy. It is true that if the signs of congestive cardiac defeat or gross pulmonary congestion show no response to medical therapy, the operative mortality is considerably increased. However, some of our most gratifying results have been in patients who at the time of operation were virtually moribund.

3. Age. In view of the high incidence of undetected rheumatic activity and because of the possibility of recrudescent activity, patients below the age of 20 are not submitted for valvotomy if operation can be delayed without risk to life. The youngest patient in this series was aged 16 at the time of operation, which was performed because of frequent exsanguinating hæmoptysis. Despite the absence of clinical and laboratory evidence of rheumatic activity left auricular biopsy showed evidence of virulent rheumatic infection.

The majority of patients submitted for mitral valvotomy have been in the fourth decade. Our oldest case was aged 55. The mortality is said to increase over the age of 50, although in a recent report the mortality for valvotomy in 35 patients between 50 and 61 years was 8.5 per cent. (Janton, et al., 1953).

Mitral Valvotomy in Pregnancy.

The operation was performed in three patients during pregnancy. It was performed in two during the sixth month as an alternative to termination by abdominal hysterotomy. Both patients suffered from tight mitral stenosis with intractable pulmonary œdema. Valvotomy was eminently successful in both. In one pregnancy continued normally and a live child was obtained; the other, however, came into labour soon after operation. A third patient, moribund because of pulmonary œdema, had an emergency valvotomy in the thirty-second week. During the operation the patient was kept almost vertical by tilting the table. Dramatic improvement was apparent immediately after the valvular split. Convalescence was uneventful apart from premature labour in the first post-operative day. The child survived. The radiological appearances before and after valvotomy are shown in Figs. 1 and 2.

Pre-operative Therapy.

Congestive heart failure is treated on the usual lines of digitalisation and diuretic therapy, till maximal improvement is obtained. In the absence of evidence of right ventricular failure digitalis is not used unless auricular fibrillation is present.

Instruction in breathing exercises is given by a competent physiotherapist for a few days before operation so that deep breathing and expectoration of mucus in the immediate post-operative period is less difficult to encourage.

Quinidine is given one hour before operation with the object of diminishing myocardial irritability.

Operative Complications.

Prolonged cardiac arrest occurred in one case in which valvotomy was done for intractable pulmonary œdema. The circulation was maintained by cardiac massage. Ventricular fibrillation incident on cardiac massage was abolished by electrical stimulation of the ventricles. Uneventful recovery followed and the patient is now, two and a half years after operation, in full employment as a motor engineer.

Three patients died from cardiac arrest, two at operation and the third two months after operation from gross cerebral damage.

Two patients with auricular fibrillation and left auricular thrombi had systemic emboli. In one a saddle embolus was removed from the aortic bifurcation two hours after operation. The other developed a hemiplegia and aphasia in the second post-operative day. Both made a good recovery. Ventricular tachycardia appeared on one occasion. This was quickly abolished by intravenous quinidine. In two cases profound hypotension occurred immediately after the valve was split. This showed an immediate response to noradrenaline given by drip and calcium chloride by injection into the left auricle.

There were two deaths from sudden circulatory failure in the immediate post-operative period. In one post-mortem demonstrated a small tear of doubtful significance in the anterior cusp of the mitral valve.

Post-operative Auricular Fibrillation.

It was found that auricular fibrillation, if present before operation, always persisted after operation. In addition, a considerable number of patients in normal sinus rhythm before operation developed auricular fibrillation in the immediate post-operative period. In these cases the rapid heart action was controlled by digitalis. It was frequently possible to establish sinus rhythm with quinidine prior to discharge from hospital. However, 15 per cent. of patients with normal sinus rhythm before operation show persistent auricular fibrillation since operation.

Results.

The operative mortality has been 5 per cent.—six deaths in 121 cases. The deaths occurred early in the series. There has been no death in the last 75 cases. Mr. J. A. W. Bingham performed the operation in five of these. In 57 cases operation was performed more than one year ago, in 25 more than two years ago. Four patients have died since operation from causes unrelated to valvotomy. One who had been markedly improved died two and a half years after operation from massive cerebral embolism. Another in whom separation of the valve commissures was impossible died two years after operation from pulmonary œdema complicating pregnancy. A third, unimproved by valvotomy, died fourteen months after operation from congestive cardiac failure. The fourth apparently had had a successful valvotomy but died suddenly eight months after operation while playing cricket.

The results shown below have been assessed by careful questioning regarding exercise tolerance and by comparison of the clinical, radiological, and electrocardiographic signs before and after operation.

Excellent	51	
Improved	19	
Slightly improved	8	
Not improved	12	
Operative deaths	6	
Post-operative deaths	4	
	100	
Too recent for assessment (operation within last three months)	21	
TOTAL	121	
Operative mortality—First 46 cases	13%	Total 5%
Last 75 cases	Nil	

ILLUSTRATIVE CASES.

Case 49.—W. G., a male aged 38. Admitted to hospital 24th July, 1952. Completely incapacitated because of exertional dyspnœa and recurrent hæmoptysis. There had been orthopnœa for some months. Examination showed pure mitral stenosis—a mitral diastolic murmur, slapping first heart sound, and well-marked mitral opening snap. A loud Graham Steele murmur was audible along the left sternal margin. X-ray screen (Fig. 3) showed a considerable increase in hilar vascularity, gross prominence of the pulmonary conus, some right ventricular enlargement, moderate enlargement of the left auricle and a hypoplastic aorta. Cardiac catheterization showed that the mean pressure in the main pulmonary artery was 125.5 cm. saline.

Mitral valvotomy was performed on 5th August, 1952. The valve was cartilagenous with some calcification at the medial commissure. Splitting occurred readily at both commissures. When reviewed six months after operation, he reported that the exercise tolerance was completely unimpaired. The auscultatory signs of mitral stenosis were unchanged. X-ray screening, however, showed a normal hilar vascularity and a diminution in prominence of the pulmonary conus (Fig. 4). When seen fifteen months after operation he stated that he was at full work as a dock labourer. Cardiac catheterization at this time showed, in comparison with the pre-operative state, a marked fall in pulmonary vascular pressure, the main pulmonary artery pressure at rest being 43.5 cm. saline and the pulmonary capillary venous pressure 15.5 cm. saline.

Case 68.—A. C., a female aged 30. Admitted to hospital in January, 1953, because of extreme exertional dyspnœa of two years' duration and recent marked nocturnal dyspnœa.

Examination showed pure mitral stenosis, there being a prolonged mitral diastolic murmur with presystolic accentuation, a slapping mitral first heart sound, and a clearly audible mitral opening snap. Crepitations were present at the lung bases. X-ray screening showed a gross increase in hilar vascularity, considerable right ventricular enlargement, a prominent pulmonary conus, and moderate enlargement of the left auricle. The left ventricle was not enlarged. The electrocardiogram showed a marked right ventricular hypertrophy pattern in the præcordial leads (Fig. 5). Cardiac catheterization showed the mean main pulmonary artery pressure to be 109 cm. saline and pulmonary capillary venous pressure 50.5 cm. saline.

Mitral valvotomy was performed on 30th January, 1953. Both commissures were readily split. Convalescence was uneventful. When reviewed on 31st March, 1953, she had already walked three miles. The mitral diastolic murmur had almost entirely gone. The first heart sound was, however, slapping and mitral opening snap still audible. Cardiac catheterization repeated in October, 1953, showed almost normal pulmonary vascular pressure at rest—main pulmonary artery = 25.5 cm. saline and pulmonary capillary venous pressure = 8 cm. The electrocardiogram recorded at this time showed that the right ventricular hypertrophy pattern in the præcordial leads had entirely disappeared (Fig. 6).

Assessment of the degree of improvement following valvotomy is frequently less easy than in the cases described above. Patients' subjective impressions regarding improvement vary. Some are hesitant to admit improvement. Others make exaggerated claims of well being. Thus after operation one patient rejoiced at his ability to get around better when, in fact, owing to the impossibility of gaining access to the left auricle, valvotomy had not been performed.

The auscultatory signs of mitral stenosis may be little affected by operation. Improvement in the radiological signs is in some cases disappointing. Indubitable electrocardiographic evidence of right ventricular enlargement is very frequently absent in mitral stenosis, a change in the electrocardiogram after operation is therefore uncommon. For these reasons an objective assessment of the results of a relatively recently introduced surgical procedure appeared to one of us (J.F.P.) to be advisable. Such an objective assessment is obtained by comparing the pre-operative and post-operative pulmonary vascular pressures as determined by cardiac catheterization.

This comparison has been made in 34 cases in the series. The result is shown in Table 2. It will be noted that there is a broad correlation between the clinical result and the post-operative fall in pulmonary vascular pressure. A marked fall in pulmonary pressures is seen in 20 of the 34 cases. In some cases, however, there is a disparity between the clinical impression of improvement and the objective evidence. Thus in Cases 5 and 12, classified as slightly improved, no fall in pulmonary pressures is noted. The fall in pulmonary capillary venous pressure in Case 24 is insignificant, yet the clinical result is regarded as excellent. It will also be noted that of 26 cases in which the result is regarded as excellent only 9 (35 per cent.) show post-operative resting pulmonary vascular pressures which approach normal.

Summary.

The assessment of patients for mitral valvotomy is discussed. The operation has been performed in 121 cases with an operative mortality of 5 per cent. Seventy per cent. of patients who have been followed up for more than three months show a worthwhile clinical improvement. In 51 per cent. the result is on clinical estimation excellent. There is, however, in some cases a disparity between clinical improvement and post-operative fall in pulmonary vascular pressures. Less than 35 per cent. of cases whose clinical result is regarded as excellent have a post-operative hæmodynamic state which approaches normal. It is nevertheless apparent

that the operation of mitral valvotomy will, in the majority of cases, convert high-grade mitral stenosis to a stenosis of much lesser degree.

TABLE 2.

		PRE-OPERATIVE		POST-OPERATIVE			
No.	Case	Mean P.A. Pressure	P.C.V.P.	Mean P.A. Pressure	P.C.V.P.	Duration Follow-up Month	Clinical Classification
5	R.B.	44.5	—	58	35	35	SI
7	J.N.	62.5	—	54	—	34	I
8	J.W.	41.5	—	27.5	—	33	I
12	G.W.	41.5 (76)	—	40.5	21	32	SI
15	M.McK.	41 (70.5)	—	31 (54.5)	21.5	29	E
17	M.D.	43.5 (62.5)	28	31.5	13	29	E
19	K.McM.	51.5 (86.5)	35.5	41 (63)	21.5	27	SI
22	M.McI.	69	66	36 (48)	25.5	25	E
23	M.B.T.	93	89.5	39	18	24	E
24	K.K.	35.5 (66)	23 (38.5)	26 (42)	19	24	E
26	J.B.	71.5 (100)	48.5	29 (42)	14	24	E
28	J.S.	48 (70.5)	31.5	25 (34.5)	10.5	23	E
30	J.M.	88.5	58	35.5	24.5	23	E
31	M.M.	54 (83)	38	23.5 (51.5)	16.5	22	E
32	A.F.	110	—	48.5	15	22	E
33	M.McM.	43 (78)	35	29	20.5	21	E
34	D.McC.	80.5 (98)	40	25.5 (41.5)	4	21	E
36	W.C.	72	—	36.5	16	20	E
38	H.S.	88.6	60	30 (35)	10	19	E
39	A.S.	52.5	—	33.5	16	18	E
41	J.M.	49	25	32.5	16	18	I
47	S.M.	54	45.5	26.5 (35)	15	18	E
49	W.G.	125.5	—	43.5	15.5	15	E
54	J.S.	43.5 (73)	34.5	32.5	21.5	13	E
58	E.S.	52.5	44	34.5	30	12	I
63	M.McD.	45	35	27	19.5	11	E
65	M.B.	37	26.5	22 (30.5)	11	11	E
68	A.C.	109	50.5	25.5	8	11	E
78	H.R.	56 (90)	34.5	24 (48)	13	8	E
79	L.C.	59	40	15.5	5	8	E
81	R.P.	71	45	43.5	28	7	E
87	C.F.	106	45	36.5	10	6	E
91	A.D.	49.5	38.5	37.5	25.5	6	I
94	Y.M.	51	34.5	23	10.5	6	E

P.A. = Pulmonary artery. Normal maximum mean pressure—20 cm. saline.
P.C.V.P. = Pulmonary capillary venous pressure. Normal maximum—12 cm. saline.
SI = Slightly improved.
I = Improved.
E = Excellent.

Mean pressures were recorded, with a saline manometer, at rest and in some cases after two minutes' exercise. The figures in brackets ( ) indicate the pressure on exercise. The reference point was the sternal angle.

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REVIEW

MEDICINE: The Patient and His Disease. By A. E. Clark-Kennedy, M.D., F.R.C.P. Volume 1. Second Edition. (Pp. 410 + xiv. 25s.). Edinburgh: E. and S. Livingstone. 1953.

IN this unusual book Dr. Clark-Kennedy has undertaken a formidable task which may be expressed as the integration of the normal physiological and psychological reactions of human beings with the many variations of both sets of reactions which occur in the presence of disease. This is the second edition and may be said to represent his maturing views on a subject which has absorbed his interest during a lifetime of study and teaching of physiology and medicine. The titles of the chapters indicate how widespread is his canvas. Chapter 1, on Body and Mind, is subdivided into sections on Energy and Matter, Life, Organic Evolution, Heredity, Development, Constitution, Consciousness and Mind. The succeeding chapters present to us a wide and humane consideration of Symptoms, Symptoms and Signs, Heredity and Environment, Reactions of Body and Mind, and finally The Nature of Disease, each divided into appropriate sections. In such a work as this there are bound to be certain uneven phases of emphasis and each reader will have his own ideas about where the stresses should have been placed; but its outstanding value is that it presents Medicine as the greatest of the "Humanities"—the proper study of mankind. The patient is more to this physician than is his disease. Here you will not find sick human beings described as clinical material nor is it implied that all the ills that human flesh is heir to are likely to be expressed in mille-equivalents. Dr. Clark-Kennedy expresses his thanks to various friends and colleagues who have helped him. Among them the present reviewer is glad to see the name of his former house physician, Professor J. D. Boyd; but this is not the only reason why he recommends this interesting book.

R. M.